

ZEBRAFISH FETAL ALCOHOL SYNDROME: EFFECTS OF ETHANOL ON MICRO-TUBULES

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Fetal alcohol syndrome is the most frequent preventable birth defect, resulting from excessive maternal alcohol consumption during pregnancy. Previous studies showed that a variety of species, including humans, exhibit developmental abnormalities when embryos are exposed to ethanol. Zebrafish embryos treated with a pathophysiological concentration of ethanol (100 mM) causes a range of defects that recapitulate some developmental defects seen in fetal alcohol syndrome (FAS) patients. The Marrs lab and others previously showed that zebrafish embryos exposed to ethanol show reduced epiboly cell movements in early embryogenesis. Microtubule cytoskeleton, especially within the large yolk cell of the zebrafish embryo, participates in the epiboly process. To understand these epiboly defects, the effect of ethanol on embryonic microtubules was investigated. Zebrafish embryos at the 2hr cell stage were exposed to 100 mM ethanol or control media for various times, fixed, and then, stained using anti-tubulin antibodies. Embryos were also stained to detect E-cadherin, actin cytoskeleton, and nuclei. These experiments showed that ethanol induced tubulin cytoskeleton redistribution in the yolk cell, which was associated with E-cadherin redistribution. Despite the redistribution of the tubulin cytoskeleton, we did not detect large differences in the microtubule staining intensity, indicating that the microtubule cytoskeleton redistributes without significant fluctuation in the amount of microtubule filament. Microtubule cytoskeleton and E-cadherin defects may contribute to epiboly defects observed in the early embryo.